Nutrition in Acute Kidney Injury (AKI)

By

Dr Olfat Abdelhalim

Clinical Nutrition Consultant

Outline

- Test your knowledge questions
- Introduction
- Metabolic abnormalities in AKI
- Nutritional requirements in AKI
  - Energy
  - Protein
  - Electrolytes
  - Vitamins
  - Fluids

1- Which of the following metabolic alterations is most commonly observed in AKI?

A- Decreased energy expenditure

B- Metabolic acidosis

C- Decreased serum Mg concentration

D- Metabolic alkalosis

2- In AKI, tight glycemic control 90-110 is highly recommended:

A- Yes, tight glycemic control proved to improve the outcome as well as survival

B- Yes, as the risk of hypoglycemia is low among patients with AKI

C- No, as the risk of hypoglycemia is high in this group of patients

D- No, tight glycemic control proved to increase the mortality in all critically ill patients including AKI

Introduction

- Clinical presentation of a patient with AKI may range from uncomplicated mono-organ failure in a non-catabolic patient to a critically ill patient with multiple-organ dysfunction syndrome (MODS).
- Metabolic changes will be determined not only by ARF per se but also the underlying disease process

Acute Kidney Injury (AKI)

Definition:

Abrupt reduction (within 48 hours) in kidney function with:

- Increase in serum creatinine ≥ 0.3 mg/dL
- Increase in serum creatinine of > 50% (1.5-fold from baseline)
- Oliguria or a reduction in urine output (< 0.5 mL/kg/hr for > 6 hrs)

- Mostly occur as a part of multi-organ failure & is associated with increased morbidity and mortality in the ICU.

(Kellum *et al. Critical Care 2013, 17:204*)

Classification of AKI

Stage 1: one of the following:

- Serum creatinine increased 1.5–1.9 times baseline
- Serum creatinine increase > 0.3mg/dl
- Urinary output < 0.5 ml/kg/h > 6 hours

Stage 2: one of the following

- Serum creatinine increase 2.0–2.9 times baseline
- Urinary output < 0.5ml/kg/h during > 12 hours

Stage 3: one of the following:

- Serum creatinine increase > 3 times baseline
- Serum creatinine increases to > 4.0mg/dl
- Initiation of renal replacement therapy
- Urinary output < 0.3ml/kg/h > 24 hours
- Anuria for more than 12 hours

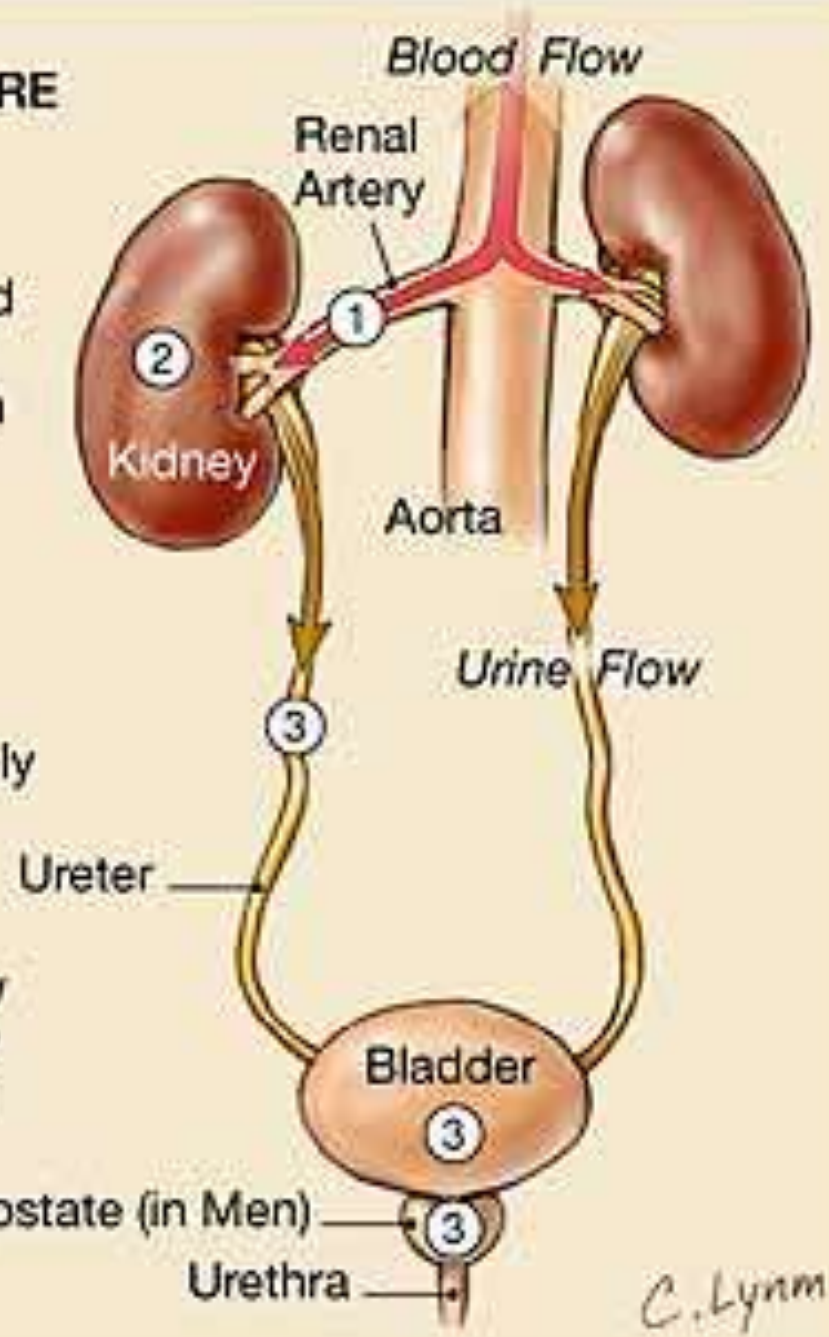
(KIDIGO Guidelines 2012)

CAUSES OF ACUTE RENAL FAILURE

① **Prerenal**
Sudden and severe drop in blood pressure (shock) or interruption of blood flow to the kidneys from severe injury or illness

② **Intrarenal**
Direct damage to the kidneys by inflammation, toxins, drugs, infection, or reduced blood supply

③ **Postrenal**
Sudden obstruction of urine flow due to enlarged prostate, kidney stones, bladder tumor, or injury



NUTRIENT METABOLISM IN AKI

- AKI associated with critical illness or injury is a ***hypercatabolic*** state that does not permit protein sparing, even when full calories are provided.
- Many studies demonstrated that provision of calories and protein in sepsis, trauma and burns stimulates whole body protein synthesis without significantly increasing whole body protein catabolism

- Metabolic disturbances specific to AKI can exacerbate the catabolism of critical illness:
  - Acidosis increases muscle breakdown
  - RRT ⟹ loss of protein (2–3 g /session)
- Decreased protein intake resulted in more muscle protein breakdown with generation of urea as those patients receiving protein moderate intake.
- Protein intake > 1.8 g protein/kg may lead to increased urea generation, and increasing calories beyond energy expenditure

Energy Metabolism and Energy Requirements

- In uncomplicated AKI, energy expenditure is within the range of healthy subjects.
- In presence of sepsis or MODS, oxygen consumption may increase by approximately 25%
- Energy expenditure in patients with AKI is rather determined by the underlying disease/associated complications and not by renal failure.

- Intake of energy substrates during nutritional support should not exceed actual energy requirements.
- Complications, if any, from slightly underfeeding are less deleterious than from over-feeding.
- Increasing energy intake increased the frequency of metabolic complications, such as hyper-glycermia and hyper-triglyceridemia, but had no beneficial effects

- Even in hypermetabolic conditions such as sepsis or MODS, energy expenditure rarely is higher than 130% of calculated basic energy expenditure and energy intake should not exceed 30 kcal/kg BW/day

Carbohydrate Metabolism

- In most AKI patients hyperglycemia is present
- **Causes:**
  - Insulin resistance
  - Accelerated hepatic gluconeogenesis
  - Abnormal insulin metabolism in AKI
  - Reduced glucose oxidation rate in AKI
- Hyperglycemia in the critically ill has been recognized as an important determinant in the evolution of complications such as infections & prognosis

Lipid Metabolism

- Profound alterations of lipid metabolism.
- ↑ LDL & VLDL ↓ Total cholesterol & HDL
- **Cause:**

  - Impaired lipolysis
- Fat particles of artificial lipid emulsions are degraded similarly to endogenous VLDL
- Fat elimination & intestinal absorption are delayed in ARF
- Elimination of I.V infused lipids is delayed in AKI (1/2 life doubled) & clearance reduced by > 50%

Protein Metabolism

- Hyper-catabolism & -ve N balance
- **Causes:**
  - Acute disease process & underlying illness
  - Acute loss of renal function
  - Type and intensity of RRT
  - Stimulation of hepatic gluconeogenesis from a.a.
  - Insulin resistance
  - Acidosis
  - Release of inflammatory mediators (TNF)
  - Depletion of antioxidative factors
  - Secretion of catabolic hormones

Electrolyte Derangements in AKI

Hyper-K

- Decreased renal elimination
- Increased release during catabolism

  (2.38 mEq/g nitrogen)

  (0.36 mEq/g glycogen)
- Decreased cellular uptake/

  increased release
- Metabolic acidosis:

  (0.6 mmol/L rise/0.1 decrease in pH)

Hyper-P

- Decreased renal elimination
- Increased release from bone
- Increased release during catabolism:

  (2 mmol/g nitrogen)
- Decreased cellular uptake/utilization
- Increased release from cells

Important Metabolic Abnormalities Induced by AKI

- Activation of protein catabolism
- Glucose intolerance/increased gluconeogenesis
- Inhibition of lipolysis and altered fat clearance
- Depletion of the antioxidant system
- Induction of a pro-inflammatory state
- Impairment of immuno-competence
- Complex endocrine abnormalities: hyper-parathyroidism, IR, erythropoietin (EPO) resistance, resistance to growth factors, etc.

Metabolic Impact of Renal Replacement Therapy

- **Amelioration of uremia intoxication Plus**
- **Heat loss (350 -700 kcal/d)**
- **Excessive load of substrates (lactate, glucose)**
- **Loss of nutrients (amino acids, vitamins)**
- **Elimination of short-chain proteins (hormones, mediators)**
- **Induction or activation of mediator cascades**
- **Stimulation of protein catabolism?**

Aim of Nutritional Support

- **The aim of MNT in AKI is:**
  - **Ensure the delivery of adequate nutrients**
  - **Prevent protein-energy wasting & its metabolic complications**
  - **Promote wound healing**
  - **Support immune system function**
  - **Reduce mortality**

Decision Making

- If a well-nourished patient can resume a normal diet within 5 days, no specific nutritional support is necessary
- In any malnourished patient, nutritional therapy should be instituted regardless of whether the patient will be likely to eat
- If the underlying diseases associated with excess protein catabolism, nutritional support should be initiated early even if the patient is likely to eat before 5 days.

- The nutritional regimen should be adapted for renal failure.
- Metabolic alteration occur in AKI when kidney function is < 30% of normal.
- Start adapted nutrition for AKI when creatinine clearance < 50 to 30 ml/min/1.73 m2 (or S. creatinine > 2.5 - 3.0 mg/dL) except in hepatic failure (↑ NH3) or MOD.

ESPEN
Recommendations

(European Journal of Clinical Nutrition and Metabolism 2010)

ESPEN Guidelines

- Energy: 20-30 kcal/kg/day
- CHO: 55- 70%
- Fat: 20-30%
- Protein: 10-15%
- Supply may range from 0.6 g/kg/day in patients on conservative treatment and up to 1.5 g/kg/day in patients on extracorporeal treatment.
- Supplementing the nutritional supply with parenteral amino acids mostly needed.

Total Caloric Provision

- Providing appropriate calories is essential to avoid excessive urea production
- Both extreme underfeeding or overfeeding appear to contribute to increased protein oxidation.
- Energy needs varies among different patients
- Energy expenditure in critically ill patients can change > 30% each day

Macronutrient Needs

- **CHO:** 3-5 g/kg/day of glucose
- **Protein:** adapted to the clinical situation and catabolic state & should be increased in patients on haemodialysis
- **Fat:** infusion should be limited to 1 g/kg/day, and lipid supply should be DC if TG levels > 300 mg/dL (usually tolerated)

Substrate Requirements in Patients with ARF

- **Energy** 25–30 (Max. 35) kcal/kg/d
- **CHO** 3–5 (Max. 7) g/kg/d
- **Lipids** 0.8–1.2 (Max. 1.5) g/ kg/d
- **Protein**
  - **Conservative therapy 0.6–0.8 (Max. 1.0) g/kg/d**
  - **Extracorporal therapy 1.0–1.3 (Max. 1.5) g/kg/d**
- **Vitamins Multivitamin preparations (vitamin C <200 mg/day)**
- **Water soluble vitamins 1–2 amp. (2 RDA) Daily**
- **Fat soluble vitamins 2–4 amp. Weekly**
- **Multi-trace-element (toxic effects) 2–4 amp Weekly**
- **Electrolytes: Individualize**

“Available evidence suggests that provision of substrates may enhance tissue regeneration, wound healing & potentially, also renal tubular repair”

“Overfeeding has well defined side effects & Complications”

Which Type of A.A Should be Used?

- Standard amino acid solutions are recommended to maintain a neutral balance.
- Non-essential amino acids have been shown to play an essential role in certain stress conditions in AKI patients.
- Only in cases where avoidance of dialysis is intended, use of solutions containing essential amino acids as the only source, at doses of 0.3-0.5 g/kg, and for < 3 weeks would be indicated.

Macro & Micronutrients Needs

- Macronutrient requirements are not so much determined by ARF as by the severity of the underlying condition, the type and intensity of extracorporeal renal replacement therapy, and the nutritional status and associated complications
- Extracorporeal treatments induce losses of micronutrients which should be supplemented & should be monitored to avoid toxic levels

- In AKI patients in ICU, electrolytes contained in EN formula providing 1,500-2,000 calories are usually adequate
- However, individual requirements may differ, and monitoring is required.
- Special care should be taken to avoid hypo-K and/or hypo-P after starting EN (re-feeding syndrome)

“In highly malnourished patients, mineral requirements may be increased due to the anabolism they experience when adequate oral diet and/or a nutritional support procedure are started”

- Assessment of vitamin A, C, and D supply is important
- At least 60-100 mg/day of vitamin C are recommended.
- Pyridoxine (5-10 mg) and folic acid (1 mg/day) are also recommended.

Which Route of Feeding is Preferable?

- Oral feeding is the most accepted route
- In patients with uncomplicated AKI, when spontaneous oral feeding is insufficient, oral supplements should be used.

EN vs. TPN

- In patients with uncomplicated AKI, EN should be used if requirements are not met with oral supplements
- EN should start early (within 24 hours)
- **Formula:** Standard formulas are the products of choice in most cases
- In case of water and electrolyte disturbances, special nephrological formulas may be useful.

- TPN is indicated when EN route is not accessible or contraindicated
- In TPN, Insulin needs will increased by nearly 25% than in EN route
- Close monitoring is recommended

NG or Jejunal Route

- The NG tube should be the access route of choice unless severe impairment in GIT motility present.

Fluids in Different AKI Phases

- Oliguric Phase
- Diuretic Phase
- In both phases fluids should be 500-800 ml/d + urine output volume
- **KDIGO 2012 guidelines:** While fluid resuscitation is widely believed to be protective, large multicenter studies have also shown that +ve fluid balance is associated with increased 60-day mortality

Volume Status

- Successful weaning from mechanical ventilation is independently associated with a negative volume status → ↑ BUN
- Management of congestive heart failure entails similar challenges to ventilator weaning in terms of balancing the needs of the kidneys with the need for diuresis.

Metabolic Acidosis

- Administration of bicarbonate as oral supplements to maintain:
  - pH > 7.2 or
  - S. bicarbonate > 17 mEq/L
- A possible dietary measure is to recommend intake of bicarbonate waters, though most patients require oral bicarbonate at different doses.

Electrolyte Intake in AKI

- **Oliguric Phase:**

  - Hyper-K: Restrict the intake to 2 g/d (30-50 mEq/d

  - Na: Restrict to ½- 1 g/d (20-40 mEq/d) depending on urine output, serum level & need for dialysis

- **Diuretic Phase:**

  - K may be needed to be supplemented

  - Na may be needed to be supplemented

There is no metabolic advantage for a protein restriction in AKI, and
moderate increases in protein intake improve nitrogen balance.

“Providing protein at 1.3–1.5 g/kg did not increase urea generation”

“Protein intake > 1.8 gm/kg
appears to increase urea generation, and may increase need for RRT”

“KIDIGO 2012 suggest administering
0.8 to 1.0 g/kg/day
protein in non-catabolic AKI patients
without need for dialysis
1.0 to 1.5 g/kg/day
in patients with AKI on RRT
up to a maximum of 1.7 g/kg/day in
patients on
CRRT and in hyper-catabolic patients”

Special Consideration

Hyperglycemia

- Another factor than can lead to increased protein catabolism and thus urea production in AKI is inadequate glucose control.
- If serum glucose is consistently >200 mg/dl, the utilization of nutrition is impaired and increased catabolism of lean muscle mass will occur.

- Glucosuria results in lost calories resulting in an increase of protein oxidation for fuel
- AKI appear to be at increased risk of hypoglycemia during intensive insulin therapy, due in part to decreased renal clearance and longer half-life of insulin
- The best is intensive insulin therapy with goal blood glucose of 140–180 mg/dl in most critically ill patients
- **KIDIGO 2012 suggest insulin therapy targeting plasma glucose 110 to 149 mg/dl**

- When intake is increased to > 5 g/kg/d, infused glucose will not be oxidized but will promote lipogenesis with fatty infiltration of the liver and excessive CO_2 production & hyper-carbia

GIT Bleeding

- Is another sources of increased urea generation
- Blood contains twice as much protein on a per-volume basis as most high nitrogen EN.

Corticosteroids

- Increased gluconeogenesis and protein catabolism, contribute to urea generation.
- Sufficient protein administration to optimize protein synthesis is advisable in those patients

Malabsorption

- Malabsorption creates a calorie deficit and thus increased oxidation of protein to meet energy needs.
- Malabsorption may occur without obvious diarrhea, especially in patients receiving narcotic pain medications that slow transit through the colon allowing increased water reabsorption.

Take Home Message

- **Acute kidney injury (AKI) in critically ill patients, seldom occurs as isolated organ failure**
- **Catabolic phase determined by critical illness, and intensified by specific derangements in substrate utilization due to the acute loss of kidney function**
- **Since AKI comprises a highly heterogeneous subjects with widely different nutrient needs along the clinical course in the same patient, nutritional requirements should be frequently reassessed, individualized and carefully integrated with RRT.**

- **In ARF the aim of nutritional treatment is not the alleviation of uremic toxicity & retardation of progression of renal disease (as in CRF)**
- **Stimulation of immuno-competence, wound healing and other reparative functions**
- **Energy 25–30 Kcal/kg/d**
- **CHO 3–5 g/kg/d**
- **Lipids 0.8–1.2 g/kg/d**
- **Protein: Conservative therapy 0.6–0.8 g/kg/d**
- **Extra-corporal therapy 1.0–1.5 g/kg/d**
- **CRRT 1.7 g/kg/d**

- **Due to the loss of the kidney's homeostatic function & disturbed metabolism accompany the critical illness**
- **AKI are especially prone to complications of nutritional support, such as hyperglycemia, hyper-TG, fluid retention, electrolyte and acid-base disturbances.**

Thank You

Case Scenario

- A male patient 56 years presented to ER by severe abdominal pain & investigations revealed abdominal aortic aneurysm for which he was operated, on day 4 post-operative he developed respiratory failure & mechanically ventillated. On day 5 the patient urine output declined & continued increase in s. creatinine

  One day after, his abdomen distended with no audible intestinal sounds & x-ray revealed air fluid level in the small bowel

- Patient weight 76 kg, height 172, BUN 42, creatinine 4.5, albumin 2.3, K 5.2
- Other lab values are within normal ranges
- Urine volume 800 ml/d
- Patient is now NPO, no plan for dialysis at this time

1- What is your nutrition plan?

2- What is needed investigations during your follow up?

"Anyone who stops learning is old, whether at twenty or eighty. Anyone who keeps learning stays young."

Henry Ford

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